beginning of the 19th century. What I said was that, on the recency hypothesis, "descriptions corresponding to adolescent insanity [in Clouston's sense of the term] will hardly be found in pre-1800 records"; and I said the earliest probable description I had found was in Cox's book of 1806 (the description is also given in his first edition of 1804). Their quotations from Haslam's books of 1809 and 1810 are therefore irrelevant as arguments against the recency hypothesis. Moreover, they fail to notice that I drew attention in my paper to the differences between Haslam's *Observations* of 1798 and his second edition of 1809. The remarkable passage in the second edition about "a form of insanity in young persons" does not occur in the first edition – that was my point. It is also noteworthy that Haslam says little about auditory hallucinations (and nothing about "voices") in his first edition (p. 5), but much more about them in his second edition (p. 68). In the second edition, but not in the first, he refers to 'the alarming increase in insanity'; and as late as 1843 he was still concerned with this increase, which he believed to be a real one (Leigh, 1961, p. 134).

Haslam's book of 1810 is entitled 'Illustrations of Madness, exhibiting a *singular* case of Insanity . . .' (my italics). Precise in his use of words and a student of etymology (Leigh, 1961, p. 116), Haslam would have chosen the word 'singular' with care. Its meaning was the same in his day (Johnson, 1807) as it is now – unique, extraordinary, unexampled, peculiar. Why then, we may wonder, should Dr Persaud and Miss Allderidge consider this case as an instance of what was 'so common, obvious and typical' as to be 'hardly worth mentioning'?

I am sure that many cogent arguments may be urged against the recency hypothesis, but I find little or nothing of substance in the criticisms of Dr Persaud and Miss Allderidge. And that leads me to think that the remarks in their final paragraph are out of place.

EDWARD HARE

47 Alleyn Road
London SE21 8AD

References

CLOUSTON, T. (1892) Developmental insanities. In *A Dictionary of Psychological Medicine*, Vol. I (ed. D. H. Tuke). London: Churchill.

COX, J. M. (1806) *Practical Observations on Insanity* (2nd edn). London: Baldwin and Murray.

CRANEFIELD, P. E. (1962) Preface to facsimile reproduction of *A Treatise on Insanity by Ph. Pinel*, trans. from the French by D. D. Davis. New York: Hafner.

HARPER, A. (1789) *A Treatise on the Real Causes and Cure of Insanity*. London: Stalker.

HASLAM, J. (1798) *Observations on Insanity*. London: Rivington.

— (1809) *Observations on Melancholy and Madness*. London: Callow.

— (1810) *Illustrations of Madness*. London: Rivington *et al.*

JOHNSON, S. (1807) *Dictionary of the English Language* (12th edn).

LEIGH, D. (1961) *The Historical Development of British Psychiatry*. Oxford: Pergamon Press.

PINEL, P. (1801) *Traite medico-philosophique sur l'alientation mental*. Trans. (1806) by D. D. Davis as *A Treatise on Insanity*. Sheffield: Cadell and Davies.

WILKINS, R. (1987) Hallucinations in children and teenagers admitted to Bethlem Royal Hospital in the nineteenth century and their possible relevance to the incidence of schizophrenia. *Journal of Child Psychology and Psychiatry*, **28**, 569–580.

Assessment of outcome

SIR: Tarrier *et al* (*Journal*, May 1989, **154**, 625–628) report a substantial rate of "relapse" of schizophrenia after 24 months of aftercare that included 9 months of a specific psychological intervention as an adjunct to drug therapy. They comment that several other recent studies of psychosocial and drug combinations have shown an increase in psychopathology once the psychosocial intervention has been completed, and advocate the continuation of such treatment for a longer period. They fail to note that the one study that did follow this valuable advice not only showed continued low levels of schizophrenic and affective psychopathology, but also showed a substantially higher recovery rate, in terms of both the clinical condition and social disability (Falloon, 1985).

The preoccupation of schizophrenia researchers with preventing "relapse", a state poorly defined in most studies (Falloon *et al*, 1983), has led to a neglect of measurements that reflect the quality of life of the patient and his caregivers. I suggest that the aim of long-term management of any chronic disorder characterised by exacerbations and remissions should focus on maximising functioning and minimising handicaps, as well as controlling symptoms. A longitudinal approach that targets the continuing and changing needs of patients and their support systems seems essential. Continued targeting of psychosocial interventions as well as drug therapies may yet lead to an enhanced rate of clinical and social recovery from schizophrenia. The advocates of such a strategy are urged to conduct further studies to assess the long-term benefits of continuing optimal multimodel therapies.

IAN R. H. FALLOON

Buckingham Mental Health Service
22 High Street
Buckingham MK18 1NU

References

FALLOON, I. R. H. (1985) *Family Management of Schizophrenia: a Study of Clinical, Social, Family, and Economic Benefits*. Baltimore: Johns Hopkins University Press.

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—, MARSHALL, G. N., BOYD, J. L. *et al* (1983) Relapse in schizophrenia: a review of the concept and its definitions. *Psychological Medicine*, **7**, 469–477.

Toxicity of antidepressants

SIR: Beaumont (*Journal*, April 1989, **154**, 454–458) has contributed in an imaginative way to the debate concerning the prescription of antidepressant medication. The principal issue reviewed in his article is that of toxicity in overdose.

A number of authors have addressed the issue of calculating the relative risk of fatal poisoning associated with antidepressants, notably Leonard (1986) and Cassidy & Henry (1987). While there is no definitive way of arriving at a calculation which will precisely predict the pattern of fatalities following overdosage with antidepressant agents (since the necessary data is unavailable), these authors provide a 'fatal toxicity index' which attempts to predict the number of deaths per million prescriptions of each drug.

The results are striking, and perhaps can be illustrated even more clearly by the extrapolations below. The total number of prescriptions for antidepressant medications in 1987, in the UK, was around 7 009 000 (Pers. Comm., ICI Pharmaceuticals). We can therefore use Cassidy & Henry's formula to predict that if only a single antidepressant agent were to be administered throughout the UK, we could expect the following consequences: exclusive use of dothiepin would result in 350 deaths per annum; amitriptyline, 326; and doxepin, 219. In contrast, use of the 'newer' antidepressants would compare as follows: trazodone, 95 deaths per annum; and lofepramine, 0. Since the body of evidence available would suggest little difference in efficacy between the spectrum of antidepressant agents, the justification for using the older, more toxic antidepressants seems tenuous. Additionally, the newer compounds have superior adverse effect profiles.

In the light of current political developments, we are likely to be subjected to progressively greater pressure to 'rationalise' our prescribing habits. Our fear is that this will involve the production of Regional Health Board and hospital formularies which will attempt to restrict our antidepressant prescribing to that of the cheapest products available. Already, in the Grampian area, we are being urged to use amitriptyline, imipramine, and doxepin.

We agree that there seems little need to use other than a small fraction of the antidepressant medications available to us. However, we suggest that restricting our clinical freedom to the use of lofepramine, trazodone, one of the serotonin re-uptake blockers, and a single MAOI (for example phenelzine) might substantially reduce the 'human cost' of our prescribing.

We are faced with balancing financial considerations against those of fatality in overdose. Since doctors are generally poor at predicting suicide attempts (Barraclough *et al*, 1974), it is currently inevitable that our patients will continue to kill themselves with the tools which we supply. Which is the greater cost?

KEITH MATTHEWS

Kingseat Hospital
Newmachar

JOHN M. EAGLES

The Ross Clinic
Cornhill Road
Aberdeen AB9 2ZF

References

BARRACLOUGH, B., BUNCH, J., NELSON, B., *et al* (1974) A hundred cases of suicide: clinical aspects. *British Journal of Psychiatry*, **125**, 355–373.

CASSIDY, S. & HENRY, J. (1987) Fatal toxicity of antidepressant drugs in overdosage. *British Medical Journal*, **295**, 1021–1024.

LEONARD, B. E. (1986) Toxicity of antidepressants. *Lancet*, *ii*, 1105.

Toxicity of hospital water?

SIR: We have recently reported (King & Birch, 1989) that toxic concentrations of heavy metals in drinking water may occur at Victorian mental hospitals with independent water supplies. The problem is compounded by compulsive water drinking among chronically psychotic patients, which is often difficult to detect and is probably under-diagnosed (Noonan & Ananth, 1977; Lee *et al*, 1989). Heavy metal neurotoxicity masquerading as psychiatric illness seems a real possibility in some of our institutions, especially where maintenance has been neglected.

We are unsure how widespread such hazards are in practice, and should be interested in hearing from anyone working in older hospitals where discoloured water may indicate corroded pipework.

J. R. KING
N. J. BIRCH

Barnsley Hall Hospital
Bromsgrove
Worcestershire B61 7AX

References

KING, J. R. & BIRCH, N. J. (1989) Health hazards on tap. *Lancet*, *i*, 1080.

LEE, S., CHOW, C. C. & KOO, L. C. L. (1989) Altered state of consciousness in a compulsive water drinker. *British Journal of Psychiatry*, **154**, 556–558.

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